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*Gong-Xin Yu, Ed Braun,
and Roger P. Wise. 2001.
Rds and Rih Mediate
Hypersensitive Cell Death
Independent of Gene-for-
Gene Resistance to the Oat
Crown Rust Pathogen,
Puccinia coronata f. sp.
avenae. **Molecular Plant
Microbe Interactions**
14(12):1376-1383.*



iseases caused by fungal pathogens are one of the greatest deterrents to grain cereal production world-

wide. An economical way of preventing pathogen-induced crop loss

is to use genetic varieties that are resistant to these pathogens. In oats, hypersensitive cell death caused by hypersensitive response (HR) at the site of infection is a significant component of defense against the fungal pathogen, *Puccinia coronata*, the causal agent of oat crown rust. HR has been proposed to play an important role in disease resistance in that cell death is thought to deprive the pathogen of nutrients, resulting in pathogen containment.

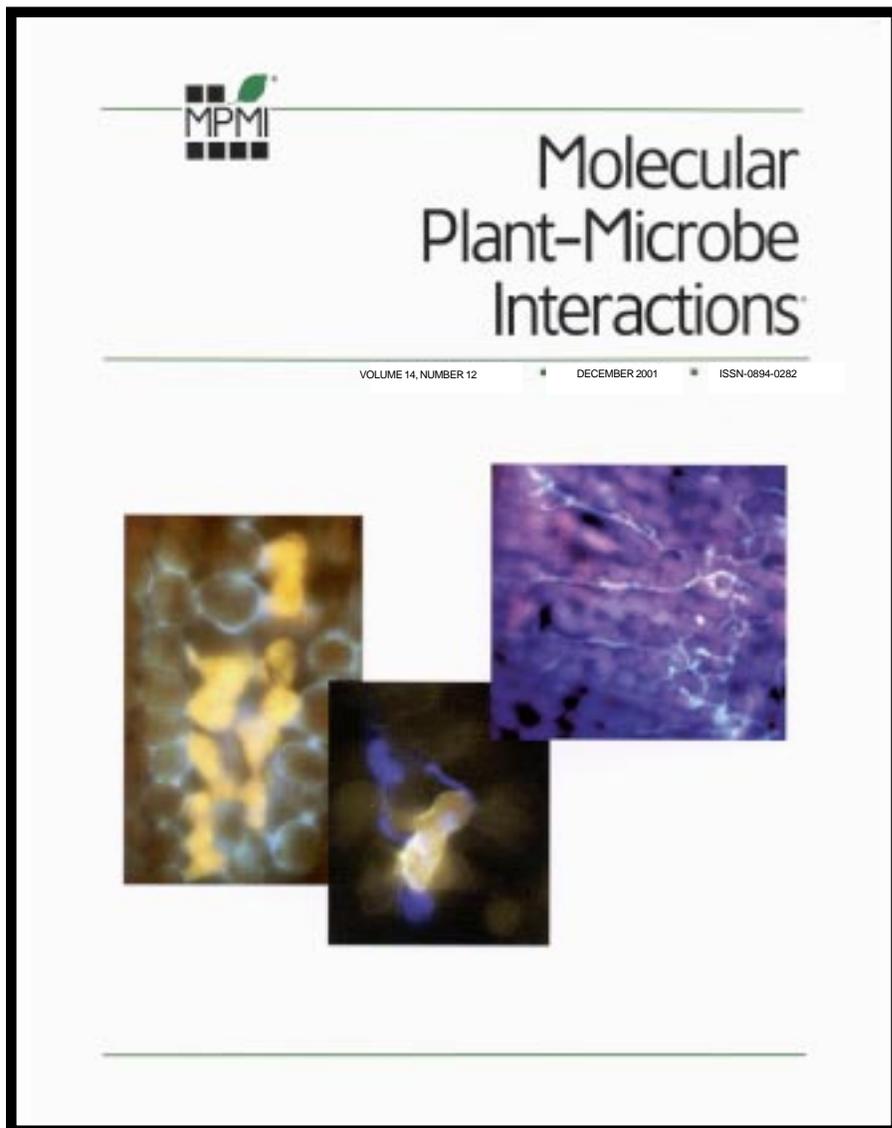
Through NRI-sponsored studies conducted by USDA's Agricultural Research Service in cooperation with Iowa State University, researchers have provided a better understanding of the interactions between pathogens and their host crop plants. Two new oat genes that regulate hypersensitive cell death were identified. The *Rds* gene suppresses the hypersensitive response, but not the resistance, mediated by the *Pc82* resistance gene. In contrast, the *Rih* gene confers the hypersensitive response in both resistant and susceptible plants. This research demonstrates that hypersensitive cell death is not essential for resistance to crown rust.

This novel description of the separation of fungal disease resistance from hypersensitive cell death illustrates the need for continued research on disease interactions in plants. Such research will ultimately lead to better plant health and increased agricultural productivity.

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